LETTERS TO THE EDITOR

A non-Hodgkin's lymphoma in a patient with HIV-2 infection

Non-Hodgkin's lymphoma is a well known complication in persons with HIV-1 infection. HIV-2 is a less pathogenic virus, but HIV-2 infected individuals also may progress to AIDS, clinically indistinguishable from AIDS caused by HIV-1 infection.[1] So far only four cases of non-Hodgkin's lymphoma in HIV-2 infected individuals have been reported.[2-5] We describe a fifth case.

A 67 year old bisexual man was diagnosed with HIV-2 infection in 1987. His CD4 lymphocyte count at that time was 216/mm.[3] In December 1988 zidovudine treatment was started. In 1989 he developed polyneuropathy and minor cognitive disorders. After 1992 he suffered recurrent attacks of angina pectoris because of severe coronary insufficiency. In December 1993 he was hospitalised because of severe weight loss, fatigue, nausea and abdominal pain, mainly localised in the left part of the abdomen. His CD4 count was 100/mm.[3] At colonoscopy a tumour was observed at the level of the splenic angle. A biopsy revealed a non-Hodgkin's lymphoma. No other lesions outside the gastro-intestinal track were found. Initially, he was treated with endoxan, adriamycin, vincristine and prednisolone. His abdominal pain disappeared but he continued to lose weight. Despite chemotherapy he developed a large bowel obstruction and died in September 1994. A post-mortem examination was not performed.

HIV-2 infection is mainly prevalent in West Africa. Because in this part of the world the facilities for the diagnosis of lymphoma are often lacking, it is likely that lymphoma remains often undiagnosed in African AIDS patients. With increasing numbers of patients with HIV-2 infection seen in the developed world it is expected that cases of non-Hodgkin's lymphoma associated with HIV-2 infection will also increase.

R COLEBUNDERS
H De VUYST
T VERSTRAETEN
Institute of Tropical Medicine, Antwerp, Belgium
W SCHROYENS
E VAN MARCK
University Hospital Antwerp, Belgium

Correspondence to: Dr R Colebunders, Institute of Tropical Medicine, Nationalestraat 155 2000 Antwerp, Belgium.

1 De Cock KM, Brun-Vézinet F. HIV-1 and HIV-2 infections and AIDS in West Africa. *AIDS* 1991;5:S89-S95.
2 Ancelle R, Bletry O, Baglin AC, Brun-Vézinet F, Rey MA, Godeau P. Long incubation period for HIV-II infection. *Lancet* 1987;i:688–9.
3 Clavel F, Manshinho K, Chamaret S, *et al.* Human immunodeficiency virus type 2 infection associated with AIDS in West Africa. *N Engl J Med* 1987;316:1180–5.
4 Lucas SB, Hounnou A, Peacock CS, *et al.* The mortality and pathology of HIV disease in a West African city. *AIDS* 1993;7:1569–79.
5 Mabey DCW, Tedder RS, Hughes ASB, *et al.* Human retroviral infections in the Gambia: prevalence and clinical features. *BMJ* 1988;296:83–6.

Accepted for publication 2 December 1994

Prurigo nodularis in an HIV positive man

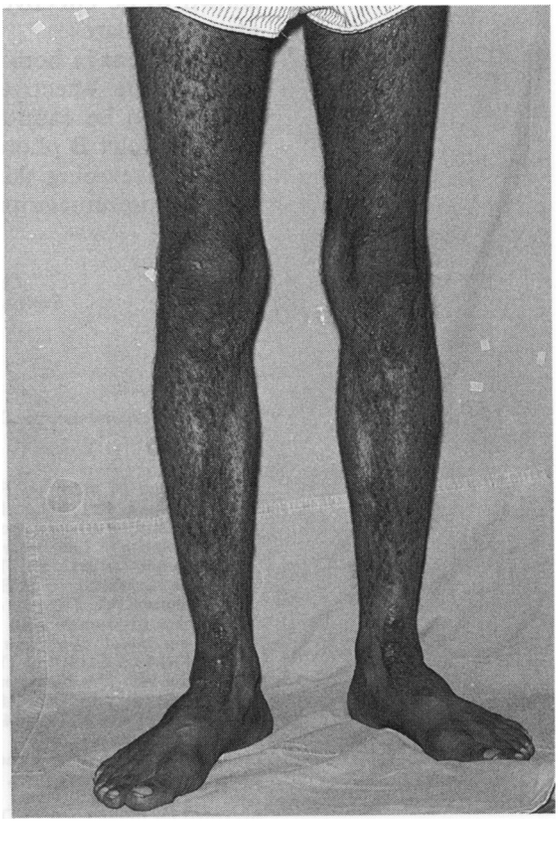
Prurigo nodularis is usually seen in atopic subjects. Recently a similar eruption has been described in HIV infection. We describe a case in which nodular prurigo was the presenting feature of HIV infection, and suggest possible treatment modalities.

A 38 year old Asian man complained of generalised pruritus for a few months prior to his presentation at the dermatology clinic. During the course of investigations he was subsequently found to be HIV positive. Examination showed nodules and lichenified papules across the chest, down the arms with very florid lesions on his legs and particularly around the ankles (fig).

Excision biopsy of one of the nodules was performed and the histology of this showed hyperkeratosis overlying epidermal acanthosis and some dermal inflammation. This in association with the history and clinical findings led to the diagnosis of prurigo nodularis.

Although this patient obtained some benefit from the application of topical steroids under occlusion for lesions on the limbs, because of the significant involvement of the trunk it was decided to treat him with ultraviolet B phototherapy.

Prurigo nodularis is a rare disease, and is characterised by chronic intensely itchy



nodules. There may or may not be an associated eczematous eruption. The lesions classically consist of single or multiple nodules situated on the extremities, especially on the anterior surfaces of the thighs and legs. A linear arrangement of the lesion is common. Among various skin manifestations in HIV positive patients, prurigo nodularis was observed in 1·8% and 6·5% of patients in two studies.[1 2]

The cause of prurigo nodularis is unknown although emotional stress can be a contributory factor, as seen in some apparently healthy HIV negative subjects. Around 80% of patients (non HIV positive) are atopic, although, often, there is no eczematous eruption present. In the remaining 20%, the condition is said to start after an insect bite. No such precipitating factors were found in the case presented. The classical symptoms of intense pruritus and nodules were present. New nodules may develop from time to time. Nodules may remain pruritic indefinitely although some may regress spontaneously to leave scars.

The large, more or less symmetrical nodules along with the history associated with the pruritus, established the diagnosis. The histology findings are considered to be consistent with prurigo nodularis but are by no means diagnostic.

Treatment of this condition can be by local application of steroids, ultraviolet phototherapy, Psoralen photochemotherapy, benoxaprofen, thalidomide, and direct injection of the nodules with a steroid can often be helpful. The latter was not attempted in the case as the lesions were too widespread, making it technically difficult to perform. In our case, the patient was treated using ultraviolet B phototherapy, which has previously been shown to be effective in pruritic HIV-associated dermatoses.[3] This resulted in an improvement in both his signs and symptoms. Despite the effectiveness of the treatment, one should be cautious about prolonged use of ultra violet B phototherapy, because of the risk of developing skin malignancies, particularly in immunocompromised patients.

ARABINDA KUNDU
A A H WADE
Department of GU Medicine, Coventry and Warwickshire Hospital, Stoney Stanton Road, Coventry CV1 4FH
A ILCHYSHYN
Department of Dermatology, Walsgrave Hospital, Coventry CV2 2DX

Address correspondence to: Dr A Kundu.

1 Wambugu P, Plummer FA, Anzala AO, Gilks C, Bosire M, Ngugi EN. Clinical manifestations of HIV-1 infection among women working as prostitutes in Nairobi. *International Conference on AIDS* 1990 20–23 June; **6(1)**:260 (abstract no Th.c.543).
2 Anzala A, Wambugu P, Bosire M, Ngugi EN; Waiyaki P, Plummer FA. The rate of development of HIV-1 related illness in women with a known duration of infection. *International Conference on AIDS* 1990 20–23 June; **6(1)**:143 (abstract no Th.c.37).
3 Penney NS. Pruritic papular and follicular eruptions in: Marks R ed, *Skin Manifestations of AIDS*: London Martin Dunitz 1990; 143.

Presented as a poster at a meeting of the Medical Society of Venereal Diseases, Liverpool, May 1994.

Accepted for publication 25 October 1994

Pancreatitis associated with aerosolised pentamidine

Pentamidine is widely used for both prophylaxis and treatment of *Pneumocystis carinii* pneumonia (PCP) and pancreatitis is a recognised adverse effect of this agent. This is generally seen during treatment of established PCP[1–5] and may occasionally be fatal.[6 7] We describe a case of acute pancreatitis following the use of monthly aerosolised pentamidine for prophylactic purposes.

A 29 year old man found to be infected with human immunodeficiency virus (HIV) in 1987 was started on co-trimoxazole for primary PCP prophylaxis in September 1991 as his absolute CD4+ cell count fell to 200×10^6/l. In March 1993 this had to be discontinued on account of severe neutropenia ($0{\cdot}37 \times 10^9$/l). He was then prescribed nebulised pentamidine 300 mg once a month. His CD4+ cell count had dropped to 10×10^6/l by March 1994 but he remained otherwise well with no AIDS defining illness. He was also unable to tolerate zidovudine but required long term acyclovir for recurrent herpes simplex and fluconazole for oral candidiasis. In June 1994, he was admitted to hospital with severe upper abdominal pain associated with nausea six days after his monthly pentamidine.

Clinically, it was felt that he had acute pancreatitis with no signs of any disseminated opportunistic infection. Serum amylase was 1116 U/l (normal range 0–95). Blood glucose, renal function and liver enzymes were normal. Serum calcium 2·13 mmol/l (normal range 2·26–2·60). Blood cultures were sterile. An abdominal ultrasound scan revealed a very oedematous pancreas with surrounding fluid but no gall stones. Abdominal CT did not reveal any neoplastic or other pathology. CT of the head (done as he complained of headaches at one stage) was also normal. His symptoms settled down with conservative treatment, the serum amylase fell to 235 U/l and he was allowed home a week later.

Hart[8] reported the case of a man who received a 21 day course of intravenous pentamidine for PCP. He was subsequently given biweekly nebulised pentamidine 60 mg and a year later developed acute pancreatitis. Prior exposure to sulpha-containing drugs has been recognised as a risk factor[2] but to our knowledge, this is the first reported case of pancreatitis due to aerosolised pentamidine when used for primary PCP prophylaxis in a relatively low dose in an individual never exposed to the agent in a therapeutic dose intravenously or otherwise in the past.

G SINGH
S M EL-GADI
R A SPARKS
Department of Genitourinary Medicine Cardiff Royal Infirmary Newport Road, Cardiff CF2 1SZ, UK

1 Murphey SA, Josephs AS. Acute pancreatitis associated with pentamidine therapy. *Arch Intern Med* 1981;141: 56–8.
2 O'Neill MG, Selub SE, Hak LJ. Pancreatitis during pentamidine therapy in patients with AIDS. *Clinical Pharmacy* 1991;10:56–8.